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Fabrikant, J.I.

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Radon and Lung Cancer: The BEIR IV Report

J.I. Fabrikant

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RADON AND LUNG CANCER: THE BEIR IV REPORT ¹

Jacob I. Fabrikant, M.D., Ph.D. ^{2,3}

Donner Laboratory and Donner Pavilion.

Research Medicine / Radiation Biophysics, Lawrence Berkeley Laboratory

University of California, Berkeley

Berkeley, California 94720

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² Professor of Radiology, University of California, Berkeley and San Francisco

³ Dr. Fabrikant was Chairman of the BEIR IV Committee, which authored the BEIR IV Report, "Health effects of radon and other internally deposited alpha-emitters: BEIR IV" (National Research Council, 1988).

ABSTRACT

The National Academy of Sciences' BEIR IV Report (National Research Council, 1988) deals primarily with the lung cancer risks in human populations exposed to internally-deposited alpha-emitting radon and its decay products. Quantitative risk estimates for lung cancer are derived from analyses of epidemiologic data. A modified excess relative risk model of lung cancer mortality of worker exposure to radon progeny in underground miners is developed; this models the excess risk per WLM (working level month) in terms of time intervals prior to an attained age, and is dependent on time since exposure and age at risk. Risk projections for the general public in indoor domestic environments are presented and cover exposure situations of current public health concern. For example, lifetime exposure to 1 WLM y^{-1} is estimated to increase the number of deaths due to lung cancer by a factor of about 1.5 over the current rate for both males and females in a population having the current prevalence of cigarette-smoking. Occupational exposure to 4 WLM y^{-1} from ages 20 y to 40 y is projected to increase lung cancer deaths by a factor of 1.6 over the current rate of this age cohort in the general population. In all of these cases, most of the increased risk occurs to smokers for whom the risk is up to ten times greater than for non-smokers. Discussion includes the extrapolation of estimates of lung cancer mortality risks from the underground miner data to the general population exposed to radon and its decay products in the indoor domestic environment.

INTRODUCTION

The terrestrial radionuclide of increasing importance to public health is radon-222, a noble gas and a decay product of radium-226 in the uranium-238 series (Nazaroff et al, 1988; Crawford-Brown, 1987; Nero, 1988; Nero et al, 1986; Nuclear Energy Agency, 1983; Eicholz, 1987; National Research Council, 1988). This gas emanates from the soil, which is the origin of the high levels of radon progeny observed in homes, and also from building materials of terrestrial

origin. It seeps into homes and office buildings and when ventilation is restricted, may accumulate in concentrations substantially higher than those prevailing outdoors. In response to the recent need to conserve energy in the heating of homes and office buildings, construction methods that sharply restrict ventilation have been introduced. As a result, the control of radon levels in indoor air has become increasingly important (Nero, 1988; USEPA, 1989; NCRP, 1989).

Within the soil, radon-222 concentrations can exceed $37,000 \text{ Bq m}^{-3}$ ($1,000 \text{ pCi L}^{-1}$). Outdoor concentrations vary considerably, but average about 7.4 Bq m^{-3} (0.2 pCi L^{-1}) with much higher concentrations at ground level. The major pathway of exposure of members of the general public is through exposure indoors, where on the average 70-80 percent of the time is spent. Because closed structures do not allow for extensive mixing of air, the concentrations of radon in buildings tend to be higher than outdoor concentrations. Indoor concentrations of radon-222 in the United States are only moderately higher, averaging about 55 Bq m^{-3} (1.5 pCi L^{-1}) and up to 300 Bq m^{-3} (8 pCi L^{-1}) or more, when ventilation is not greatly restricted (Nero et al, 1986). These indoor radon concentrations can vary widely from the ambient air outdoor value to values that are a few thousand times higher (NCRP, 1989). Measurements show an apparent log-normal distribution of concentrations in indoor air, based on surveys in homes. About 2 percent of U.S. homes exceed levels of 300 Bq m^{-3} (8 pCi L^{-1}).

The tissues at risk from exposure to radon and its progeny include the epithelium of the bronchi, segmental bronchioles, and alveolar membranes (National Research Council, 1988; Cross, 1987; Eicholz, 1987; James, 1988; James et al, 1987). The most important tissue is the bronchial epithelium, which is the site of most lung cancers thought to be induced by radiation. These tissues are exposed primarily to radon daughters, e.g., polonium-218, which attach themselves to dust particles and, when inhaled, deposit themselves within the respiratory system at locations influenced by particle size. The epithelium of alveoli receives an estimated dose equivalent of approximately 5 mSv y^{-1} (0.5 rem y^{-1}) when radon concentrations in air are 37 Bq

m^{-3} (1 pCi L^{-1}). The dose equivalent to the segmental bronchioles may be approximately 5 times higher. For smokers, the additional exposure to the lungs from plutonium-210 volatilized from smoking tobacco increases dose equivalent to the bronchial epithelium considerably (National Research Council, 1988). Furthermore, the epithelium of the segmental and subsegmental airways is the principal site from which most cigarette-smoking-related lung cancers arise.

DOSIMETRY

By convention, the concentration of radon progeny is measured in working levels (WL), and cumulative exposures over time are measured in working-level-months (National Research Council, 1988; cf. Chap. 2 and Annex 2B). The working level (WL) is defined as any combination of short-lived radon daughters in 1 liter of air that results in an ultimate release of 1.3×10^5 MeV of potential alpha energy. This is approximately the amount of alpha energy emitted by the short-half-life radon daughters in equilibrium with 3,700 Bq (100 pCi) of radon. Exposure of a miner to this concentration for a working month of 170 h (or twice this concentration for half as long, etc.) is defined as a working-level month (WLM). The cumulative exposure in WLM is the sum of the products of radon-progeny concentrations and the times of exposure.

The relationship between exposure, measured as WLM, and dose to target cells and tissues in the respiratory tract is extremely complex and depends on biologic, physical and chemical factors (NCRP, 1984). Because of differences in the exposure conditions, it cannot be assumed a priori that exposure to 1 WLM in a home and to 1 WLM in a mine will result in the same dose of alpha radiation to cells in the target tissues of the respiratory tract. An understanding of the dosimetry of radon daughters in the respiratory tract is essential in extrapolating lung cancer risk estimates derived from epidemiologic studies in miners to the general population in indoor domestic environments. Three broad factors influence the dosimetry of radon daughters---the

physical characteristics of the inhaled air, the breathing patterns, and the physiologic-anatomic characteristics of the lung (National Research Council, 1988; cf. Chap. 2 and Annex 2B).

Radon daughters are initially formed as condensation nuclei. Although most of these particles attach to aerosols immediately after formation, a variable portion remain unattached. The unattached fraction is an important determinant of the dose received by the target cells in the bronchial epithelium; as this fraction increases, the dose also increases because of the efficient deposition of the unattached daughter in the airways of the lower respiratory tract. The particle size distribution in the inhaled air, i.e. the aerosol characteristics, also influences the dose to the airways, because particles of different sizes deposit preferentially in different generations or regions of the lung airways. The specific mixture of radon daughters, i.e., the equilibrium of radon with its daughters, also affects the dose to the target cells, but to a lesser extent (National Research Council, 1988; cf. Chap. 2, Annex 2BB). Crawford-Brown, 1987; James, 1980; James et al, 1988; cf. Chap. 2, Annex 2B; Nero, 1988).

Breathing patterns are extremely important. The amount of radon daughters varies directly with the minute ventilation, i.e., the total volume of air inhaled in each minute. The deposition of radon daughters within the lungs, however, does not depend in a simple fashion on the minute ventilation, but rather varies with the flow rates in each airway generation (i.e., bronchi, bronchioles, etc.). The flow rates vary with both tidal volume and breathing frequency. The proportion of oral and nasal breathing also influence the relationships between exposure and dose. A large fraction of the unattached radon daughters deposits in the nose with nasal breathing, whereas it is likely that a smaller fraction deposits in the mouth with oral breathing (ICRP, 1979; ICRP, 1987; Jacobi and Eisfeld, 1980; James, 1988; James et al, 1980; NCRP, 1984; National Research Council, 1988; Nero, 1988).

The anatomic and physiologic characteristics of the lung influence the relationship between exposure and dose. The sizes and branching patterns of the airways of the lower respiratory tract affect deposition and can differ between males and females, and between adults and children. The rate of mucociliary clearance and the thickness of the mucous layer in the airways also influence the dose, as do the locations of the target cells in the bronchial epithelium. Smoking and other environmental pollutants modulate these factors to a considerable degree. The effects of many of these factors influencing dose to the target cells in the respiratory tract from radon exposure are not fully understood, but dosimetry of radon daughters can be estimated by computer modelling (National Research Council, 1988; cf Appendix VIII; James, 1988; ICRP, 1987; Nuclear Energy Agency, 1983; Jacobi and Eisfeld, 1980; James et al, 1980). The BEIR IV Committee utilized such computer models to provide guidance on estimating the risk of lung cancer due to radon in indoor environments (National Research Council, 1988; cf. Appendix VIII).

HUMAN POPULATIONS AT RISK

Current scientific reports (National Research Council, 1988; ICRP, 1987; NCRP, 1984; UNSCEAR, 1988; USEPA, 1989) concentrate on lung cancer risks due to exposure to radon and its progeny, primarily because of a need for a comprehensive characterization of lung cancer risk associated with exposure to radon and its short-lived daughters in indoor domestic environments. Estimation of lung cancer risk appears to be best derived from epidemiologic surveys of underground miners throughout the world who breathe widely-differing levels of radon-222 progeny (National Research Council, 1988; cf. Appendix IV; ICRP, 1987; NCRP, 1984; UNSCEAR, 1988; USEPA, 1989). Calculations based on dosimetric models of the respiratory tract are complex, and values are based largely on the location of the target cells in the bronchial epithelium, the physiologic processes involved in the variable dosimetry, and uncertainties introduced by numerous factors that modify lung cancer risk, such as smoking (ICRP, 1979; ICRP, 1987; National Research Council, 1988; cf. Chap. 2; NCRP, 1984). Furthermore, the

biologic assumptions, mathematical models, and radiation dosimetry are uncertain; following deposition of the alpha particles within the respiratory tract, the radiation exposure usually has a complex time pattern with varying distribution, and the actual doses and dose rates in the susceptible bronchial epithelium are often inadequately known (James, 1988; National Research Council, 1988; Chap. 2, Annex 2B; National Research Council, 1990). It is for these reasons that the need for guidance on radiation protection from the potential lung cancer risks of radon and its daughter products is of current and future concern. For a considerable period, such guidance has been directed primarily to those occupationally exposed, such as underground miners; we now include the general population, pregnant women, children, and persons who suffer from health conditions that might render them more sensitive to radiation injury (National Research Council, 1988 cf. Chap.2; National Research Council, 1990).

Numerous epidemiologic studies of underground miners exposed to radon daughters in the air of mines have shown an increased risk of lung cancer in comparison with nonexposed populations (UNSCEAR,1988; National Research Council; 1988; cf. Appendix IV; Cross, 1987). Laboratory animals exposed to radon daughters also develop lung cancer (National Research Council, 1988; cf. Appendix III; Cross, 1987; Cross, 1988). There is abundant epidemiologic and experimental data to establish the carcinogenicity of radon progeny. These observations are of considerable importance because uranium, from which radon and its progeny arise, is ubiquitous in the earth's crust, and radon in indoor environments can reach relatively high levels (Nero, 1988). Nevertheless, while the carcinogenicity of radon daughters is established and the hazards of high levels of exposure during mining is well recognized, the risks of exposure to lower levels of radon progeny have been incompletely characterized (Nero et al, 1986; Nero, 1988; National Research Council, 1988; cf. Chap. 2). However, risk estimates of the health effects of lower levels of exposure are nevertheless needed to address the potential lung cancer risks of radon and radon daughters in homes and to determine acceptable levels of exposure in occupational environments.

EPIDEMIOLOGIC STUDIES OF UNDERGROUND MINERS

Two approaches are currently being used to characterize the lung cancer risks of radon daughter exposure: mathematical representations of the respiratory tract that model radiation doses to target cells (NCRP, 1984; ICRP, 1987; USEPA, 1989) and epidemiologic investigation of exposed populations, mainly underground miners (ICRP, 1987; National Research Council, 1988; cf. Appendix IV; National Research Council, 1990, UNSCEAR, 1988; Steinhausler, 1988). The dosimetric approach used by a number of investigators and committees provides an estimate of lung cancer risk of radon daughter exposure that is based specifically on modeling the dose to target cells in the bronchial epithelium. A number of different dosimetric models have thus far been developed (National Research Council, 1988; cf. Appendix VIII); all require certain relevant assumptions, some not subject to direct verification, concerning the deposition of radon daughters in the respiratory tract and the type, nature and location of the target cells for cancer induction (National Research Council, 1988; cf. Appendix VI). Accordingly, the BEIR Committee (National Research Council, 1988) chose not to use dosimetric models for calculating the lung cancer risk estimates. The results of such dosimetric models were used to extrapolate lung cancer risk coefficients derived from the epidemiologic studies of occupational exposure of the underground miners to the general population in indoor environments. However, the lung cancer risk estimates for radon daughter exposure derived by the BEIR IV Committee (National Research Council, 1988; cf. Chap. 2, Annex 2A) were based solely on the epidemiologic evidence.

The available epidemiologic data of studies of underground miners exposed to radon daughters provides a direct assessment of lung cancer mortality. While each of the individual investigations has limitations, the approach of a combined analysis of four major data sets permitted the Committee to carry through a comprehensive assessment of the lung cancer risks of radon daughter exposure and of factors influencing the risk of exposure. In analyzing the data, the

Committee used a descriptive analytical approach rather than using statistical methods based on conceptual models of carcinogenesis or radiation (dose-reponse) effects (National Research Council, 1988; cf. Chap. 2, Annex 2A). Data were obtained from four principal studies of radon-exposed miners---the Ontario uranium miners (Muller, 1984; Muller et al, 1983; Muller et al, 1985), the Saskatchewan uranium miners (Howe et al, 1986), the Swedish metal miners (Radford and Renard, 1984), and the Colorado Plateau uranium miners (Hornung and Meinhardt, 1987) (Table 1), and developed risk models for lung cancer from the Committee's own analyses.

Table 1
Characteristics of the Four Underground-Miner Cohorts Analyzed (NR88)
(National Research Council, 1988; cf. Chap. 2)

Cohort	No. of Workers Followed (y)	Average Duration of Followup (y)	Average Age at End of Followup (y)	Average Duration Exposure (y)	Average Cumulative Exposure (WLM)	No. of Lung Cancer Deaths
Eldorado,	1,580 ^a	14	45	—	—	—
Beaverlodge, Saskatchewan	6,847	14	43	3.2	22	65
Ontario	570 ^a	16	52	—	—	—
	11,076	19	50	3.7	37	87
Malmberget	1,292	21	67	20.0	98	51
Colorado (all)	3,347	25	57	8.0	822	256
Colorado (≤2000 WLM)	2,975	25	57	7.0	509	157

^a surface workers

The follow-up experience of the groups analyzed totaled almost 500,000 person-years at risk and included 459 lung cancer deaths. There are important differences among the four studies including the duration and person-years of follow-up, the exposure rates, and average duration of exposures. These factors were evaluated extensively and were examined to the extent possible in the epidemiologic analyses.

TIME-SINCE-EXPOSURE RISK PROJECTION MODEL

The analyses indicated that the age at risk and the time since cessation of exposure were significant factors modifying the excess relative risk of lung cancer mortality. The BEIR IV Committee developed a time-since-exposure (TSE) model (National Research Council, 1988; cf. Chap. 2), which modeled the excess risk per WLM in terms of time intervals prior to an attained age. The modified exposure-time-response relative risk model was tested by analyzing the data for each of the underground miner cohorts separately. These analyses indicated that common values of the factors for age at risk and time since exposure could be applied to all four miner cohorts and that these factors operated largely independent of one another. As a final step, the combined epidemiologic data were re-analyzed with these values to obtain a maximum likelihood estimate of the excess lung cancer risk per WLM. The estimate of the lung cancer risk $r(a)$ from exposure to radon progeny, based on the occupational data, was obtained from the following modified relative risk, time-since-exposure model:

$$r(a) = r_0(a)[1 + 0.025\gamma(a)(W_1 + 0.5W_2)]$$

where $r(a)$ is total risk of lung cancer at age a , i.e., the age-specific lung cancer mortality rate; and $r_0(a)$ is the baseline lung cancer risk, or age specific background lung cancer mortality rate, which varies with sex, smoking status, and calendar period. The age factor ($\gamma(a)$) is 1.2 for ages <55 y, 1.0 for ages 55-64 y, and 0.4 for ages 65 y or more. W_1 is the cumulative WLM incurred between 5 and 15 y prior to age a , and W_2 is the WLM incurred 15 y or more before age a . The diminishing

risk of lung cancer mortality after age 64 y and the discounting of exposures occurring more than 14 y before age a resulted in considerably smaller estimates of lifetime risk than a constant relative risk model fitted to the same data. The value of $\gamma(a)$ is the lung cancer mortality rate from all causative agents, not just that due to radon exposure alone (National Research Council, 1988; cf. Chap. 2).

Thus, using statistical regression techniques appropriate for survival time data, the risk or probability of dying or lung cancer due to radon daughter exposure in the four combined cohorts and in the absence of smoking appeared to be best described by a complex modified multiplicative time-since-exposure statistical model which is strongly dependent on age at risk (National Research Council, 1988; Chap. 2). In this model, although simple in its mathematical formulation, the excess relative risk after a 5 y lag period varies with time since exposure rather than remaining constant and depends on age at risk. This expression, therefore, is a departure from most previous risk models which have assumed that the relative risk is constant over both age and time (ICRP, 1987; NCRP, 1984; Pierce and Preston, 1985; Mills and Egan, 1987). Radon exposures more distant in time have a somewhat lesser impact on the age-specific excess relative risk, is higher for younger persons and declines at older ages. The analysis did not assume a priori that analysis based on the relative risk model was necessarily more appropriate than alternatives, such as the absolute risk model. However, an absolute risk model would have involved a complex power function of age. The modified relative risk form provided a simpler description of observed lung cancer risks in the miner cohorts; it required fewer variables than would an absolute risk form (National Research Council, 1988; cf. Chap. 2).

LUNG CANCER RISKS

Recognition that radon and its daughter products may accumulate to high levels in homes has led to public health concern about the potential lung cancer risk in the general population

resulting from indoor domestic exposure to radon progeny in houses (USEPA, 1989; NCRP, 1989). Such lung cancer mortality risks can be estimated with the BEIR IV risk model. However, since the model was based on occupational exposure data of underground miners, several assumptions were required to transfer lung cancer risk estimates from an occupational setting to the indoor domestic environment. Accordingly, the BEIR IV Committee assumed that the epidemiologic findings in the underground miners could be extended across the entire lifespan, that cigarette smoking and exposure to radon daughters interacted multiplicatively, that exposure to radon progeny increased the risk of lung cancer proportionally to the sex-specific baseline risk, and that exposure to a WLM yielded an equivalent dose to the respiratory tract and to the bronchial epithelium in both the occupational and domestic home settings (National Research Council, 1988; cf. Chap. 2). This last assumption was a qualitative decision by the Committee; however, it was concluded that additional data on ventilation rates and aerosol characteristics in mines and homes were needed to address quantitatively the comparative dosimetry of radon daughters in the occupational and indoor domestic settings (National Research Council, 1988; cf. Chap 2).

Based on the estimates of excess relative risks per WLM of exposure to radon progeny derived from analysis of the four miner cohorts, and the assumptions outlined, the BEIR IV Committee (National Research Council, 1988; cf. Chap. 2) projected lung cancer risks for United States males and females. The lung cancer risk projections estimate lifetime risks, ratios of lifetime risk, average lifespans, and average years of life lost for various exposure rates and durations of exposure. The Report estimated risks conditional on survival and exposure to a particular age and for smokers and nonsmokers of either sex. For projecting lifetime cancer risks due to radon exposure, the Committee used the 1980-1984 United States mortality rates as referent rates, and applied a 5-y lag period.

As illustrative examples of the lung cancer risks associated with exposure patterns of current concern (National Research Council, 1988; cf. Chap. 2), for example, the current standard

for radon daughter exposure in underground mines limits the annual total to 4 WLM. The baseline average lifetime risk of lung-cancer mortality for all males is 0.067; the average lifetime risk of lung-cancer mortality for males of unspecified smoking status sustaining 4 WLM annually from age 20 through age 50 is 0.131; this is about twice the baseline risk for all males. Occupational exposure to 4 WLM y^{-1} from ages 20 y to 40 y is projected to increase lung cancer deaths in males by a factor of 1.6 over the current rate of this age cohort in the general population. If the miners are smokers, lifetime risk of 4 WLM annually for ages 20 y through 50 y is 0.226, compared with 0.123 for unexposed male smokers, or about twice the risk. On the assumption that the occupational results could be applied to radon exposures in houses, then lifetime exposure to 1 WLM y^{-1} is estimated to increase the number of deaths due to lung cancer by a factor of about 1.5 over the current rate for both males and females in a population having the current prevalence of cigarette-smoking. In the indoor domestic environment, female smokers exposed to 1 WLM annually from age 20 y through 60 y have a lifetime lung cancer risk associated with that exposure of 0.087, about 1.5 times the risk for unexposed female smokers. If it is assumed that the average U.S. male is exposed to 0.2 WLM y^{-1} at average ambient levels in the home environment (NCRP, 1984; National Research Council, 1988), then on the basis of 1980-1984 U.S. mortality rates (i.e., lung cancer mortality risk of 0.067 for males of unspecified smoking status), the lifetime lung-cancer mortality rate associated with exposure at 0.2 WLM y^{-1} is 0.074---an increase of about 10 percent. In all of these cases, most of the increased risk occurs to smokers for whom the risk is up to ten times greater than for non-smokers (National Research Council, 1988; cf. Chap. 2).

COMPARISONS OF LIFETIME RISK OF LUNG CANCER MORTALITY

Comparisons of estimates of the lifetime risk of lung cancer mortality due to a lifetime exposure to radon progeny in terms of WLM and alpha-particle dose to the target cells of the bronchial epithelium, made by the BEIR IV and other scientific committees over the past decade

yield a broad range of lung cancer risk coefficients (Table 2). In each of the studies, the epidemiologic data available, the dosimetric and statistical models applied, and the assumptions introduced, were quite different, and with differing and alternative methods of analyses. Although

Table 2
Estimates of Lifetime Risk of Lung Cancer Mortality
Due to Lifetime Exposure to Radon Progeny

(cf. National Research Council, 1990)

Study	Year	Excess Lifetime Lung Cancer Mortality (deaths/10 ⁶ person-WLM)
BEIR IV ^c	1988	350
ICRP ^d	1987	170-230 ^a
	1990 ^b	360 ^b
EPA ^{c,f}	1986,1988	115-400
	1989	360 ^g
NCRP ^h	1984	130
BEIR III ⁱ	1980	730
UNSCEAR ^{j,k}	1977	200-450
	1988	150-450

^a multiplicative risk projection model and an ICRP reference population (ICRP, 1987)

^b multiplicative risk projection model with the 1980-1984 U.S. reference population as applied by the BEIR IV Committee (National Research Council, 1988; National Research Council, 1990)

^c National Research Council, 1988

^d ICRP, 1987

^e U.S. Environmental Protection Agency, 1986

^f Puskin and Yang, 1988

^g U.S. Environmental Protection Agency, 1989

^h NCRP, 1984

ⁱ National Research Council, 1980

^j UNSCEAR, 1977

^k UNSCEAR, 1988

the BEIR IV Report (National Research Council, 1988) used much information not available for the earlier reports, the differences reflect mainly differences in assumptions made and the models used by the various committees; the BEIR IV Committee developed risk models for lung cancer mortality from its own analyses of the epidemiologic studies, i.e., the modified relative risk time-since exposure projection model was derived from a formal statistical analysis of primary data from four of the most complete epidemiologic studies of underground miners (National Research Council, 1988; cf. Chap. 2, Annex 2A).

Direct comparisons of these risk estimates and the studies from which they are derived are not possible because of the differences in the models used, the populations assumed to be at risk, e.g., duration of exposure and smoking prevalence, differences in the assumed lung cancer rates in the reference populations, and modeling of the smoking data and its interaction with alpha radiation. Table 2 of lifetime risk estimates of lung cancer mortality suggests that the range of values is fairly broad; this is largely due to the difference in models used and the reference populations used to project lifetime risks. The analyses over the past decade indicate that the BEIR IV estimate is near the middle of the range of risk estimates. It is about three times larger than the 1984 NCRP (NCRP, 1984) value, and about half of the estimate of the BEIR III Committee (National Research Council, 1980). The latter two reports assumed an additive risk model; the BEIR III Committee (National Research Council, 1980) based its projections on a model that is constant over time and on an increasing excess risk with age, while the NCRP (NCRP, 1984) projection is based on a risk model with diminishing excess risks with time after exposure. The BEIR IV Committee's (National Research Council, 1988; cf. Chap. 2) estimate is based on a time- and age-dependent modified multiplicative risk projection model that takes into account the reduced risk at age 65 or greater and the small effectiveness of exposures occurring 15 y or more in the past that were identified in the miner cohort data. The Environmental Protection Agency (USEPA, 1986; Puskin and Yang, 1988) estimate is based on a constant multiplicative risk model and a

United States reference population. The ICRP (ICRP, 1987) estimate is based on a constant multiplicative risk projection model and a European reference population; lifetable methods used by the 1988 UNSCEAR Committee (UNSCEAR, 1988) are essentially identical to those used by the 1987 ICRP Committee (ICRP, 1987). When the ICRP and BEIR IV values are calculated for the 1980 U.S. reference population, the risk estimates are almost the same, viz., 360 and 350 excess lung cancers per 10^6 person-WLM, respectively (National Research Council, 1990). The latest Environmental Protection Agency (USEPA, 1989) estimate is based on an average of the BEIR IV (National Research Council, 1988; cf. Chap. 2) and the ICRP (ICRP, 1987) values and the 1980-1984 U.S. reference population. One reason the BEIR IV Committee (National Research Council, 1988; cf. Chap. 2) risk projection model was developed with more extensive modifications than others, was that the original data from the epidemiologic studies of the miners were made available to the Committee for combined analyses, while other reports relied solely on published data that frequently lacked information on time- or age-dependent factors that modify risks (National Research Committee, 1988; cf. Chap. 2 and Appendix VIII). The UNSCEAR Committee (UNSCEAR, 1988) found no recent data or analyses that suggest any reason for a change in the previous lung cancer risk estimates of 1.5 to 4.5 fatal lung cancers per 10^4 person-WLM, i.e., in accord with that estimated by the 1988 BEIR IV Committee (National Research Council, 1988; cf. Chap. 2).

UNCERTAINTIES

The uncertainties in the data analysis that affect the estimates of the lung cancer risk due to exposure to radon progeny given in the BEIR IV Report (National Research Council, 1988; cf. Chap. 2) include sampling variation in the primary data, random and possibly systemic errors in the original data on exposure and lung cancer occurrence, inappropriate statistical models for analyses or misspecification of the components of the models, and incorrect description of the interaction between radon-daughter exposure and cigarette-smoking. In addition, the actual

computed lifetime risk and expected life shortening depend on the age-specific disease rates of the referent population; in the BEIR IV Committee's examples, it was the 1980-1984 United States population mortality rates. Projections based on a different referent population would be expected to differ, although the ratio of lifetime risks and years of life lost to ambient values may be more stable across populations (National Research Council, 1988; cf. Chap. 2).

The uncertainties in the lung cancer mortality risk projections associated with radon daughter exposure in the general population, on the other hand, required the application of a number of statistical techniques and assumptions concerning analyses of the populations at risk. The risk model was developed with data on the four groups of miners; the subjects were male, a limited age span and duration of exposure were covered, smoking was not explicitly considered in the analyses, and follow-up had not yet extended across the subjects' lifetimes. It was necessary, therefore, to make assumptions concerning a number of modifying factors, including the effects of sex, age at exposure, interaction between cigarette smoking and radon daughters, lifetime expression of lung cancer risk, and extrapolation of the lung cancer mortality risk model based on the underground mining environment to exposure conditions in the domestic indoor environment (National Research Council, 1988; cf. Chap.2).

Sex. The BEIR IV Committee's model was based only on data on males; with the exception of the small studies of indoor exposure, epidemiologic data on females exposed to radon daughters were unavailable. Accordingly, the Committee chose to make risk projections based on the assumption that the relative risk was the same for males and females. This was consistent with the apparent lack of sex-specific effects for cigarette smoking. No biologic rationale could be identified for considering that sex influences the development of radon-related lung cancer (National Research Council, 1988; cf. Chap. 2).

Age at Exposure. In the analysis of the miner data, no effect of age at first exposure was found. Exposure at an early age, particularly before the age of 20 y, might have greater effects at later ages; dosimetric models also project greater risk associated with exposure at younger ages (NCRP, 1984). In the absence of reliable data on younger miners and on persons exposed during childhood, the BEIR IV Committee (National Research Council, 1988; cf. Chap. 2) assumed that age at exposure did not affect the lung cancer risk associated with radon-daughter exposure, an assumption that appears to be supported by the miner data. This source of uncertainty would not have major consequence for lifetime projections of lung cancer risk.

Cigarette Smoking. Most of the data sets of the miner cohorts available for analysis by the BEIR IV Committee provided little or no information on several important aspects of smoking, e.g., smoking practices among the Canadian and Swedish miners, time since cessation of tobacco use and inhalation practices, and precluded precise description of the interaction between cigarette smoking and radon-daughter exposure. Based on the relevant evidence, principally on the strength of the Colorado Plateau data, a multiplicative interaction on a relative risk scale was considered appropriate for risk projections and this was consistent with the Committee's analyses of the data on the miner cohorts. However, a submultiplicative interaction was also consistent with the data analyzed. The Colorado Plateau data set was not compatible with an additive model; most of the other data sets were small and could be considered compatible with almost any model for interaction. It was concluded, that if the multiplicative model for the interaction were incorrect, then it would result in an overestimate of the risk associated with exposure to radon progeny for smokers, and more substantially, underestimate the risk for nonsmokers (National Research Council, 1988).

Temporal Expression of Risk. Because a short and limited period of observation of each of the underground miner cohorts was available, and since the Swedish study only provided near-lifetime observation of a large proportion of that cohort, lung cancer risk projections required

assumptions concerning the potential temporal expression of risk. The time-since-exposure model developed by the BEIR IV Committee (National Research Council, 1988; cf. Chap. 2) implies that the effects of radon progeny decline with age, but not to zero, regardless of the number of years since exposure. Whether the risk of radon-induced lung cancer does eventually return to the spontaneous background value, and if so, the time required for this to occur, cannot now be established; however, the modified relative-risk model used for risk projection applied to background rates varies with age and time since exposure.

Extrapolation from Mining to Indoor Environment. Dosimetric models provide a framework for assessing the difference between estimated lung doses in mines and in homes. The Committee chose to rely on the results of other investigators who have constructed dosimetric models and have provided descriptions of the effects of varying the values of parameters (e.g., characteristics of inhaled air, breathing patterns, characteristics of the lung) that differ in mines and homes. Within a range of uncertainty, the ratio of exposure-to-dose relationships in homes and mines can be considered to be quantitatively similar for risk projections; therefore, the BEIR IV Committee assumed that exposure to 1 WLM in a home and exposure to 1 WLM in a mine would have equivalent potency in causing lung cancer (National Research Council, 1988; cf. Chap. 2 and Annex 2B).

LUNG CANCER RISKS AND RADON PROGENY IN HOMES

RADON EXPOSURES IN HOMES AND LUNG CANCER RISK

Nero et al (Nero, et al, 1986; Nero, 1988) have presented a systematic appraisal of data from several surveys of home radon exposure in the United States. Their work suggests that the distribution of radon exposure rates in homes follows approximately a log-normal distribution with geometric mean of 35.5 Bq m^{-3} (0.96 pCi L^{-1}) and geometric standard deviation of 105.1 Bq m^{-3}

(2.84 pCi L⁻¹). This estimate was based on 22 sets of data from surveys which were generally carried out to ascertain potential increases in radon exposure resulting from energy conservation procedures. The shape of the distribution and the estimates of its parameters were at best only an approximation of the true levels of radon found in American homes. In addition, homes that were selected for measurement were provided by volunteers, so that the overall sample may be weighted toward housing of the middle and higher socioeconomic groups where energy conservation awareness is relatively higher.

To provide some perspective of the lung cancer risk due to radon exposure, comparisons might be made with the expected risk in the United States. An estimated 130,000 lung cancer deaths occurred in 1986; 89,000 in males and 44,000 in females. About one death in 20 is due to lung cancer, a lifetime risk of 5 per cent. It has been estimated that cigarette smoking is responsible for 85 per cent of lung cancers among men and 75 per cent among women, some 83 per cent overall. The lifetime risk of lung cancer for nonsmokers is somewhat less than 1 per cent. Even for the nonsmoker, passive smoking may contribute to this 1 per cent or less; it has been estimated that passive smoking may be a contributor to this one per cent in U.S. nonsmokers. On average, a smoker's risk is about ten times that of a nonsmoker.

The risk of radon-induced lung cancer among residents of single family homes in the United States (approximately 70 per cent of the housing stock) has been estimated using the time-since-exposure relative-risk projection models developed by the BEIR IV Committee (Lubin and Boice, 1989). These models, based on extrapolation to lower radon exposure levels from exposure-time-response relationships observed at higher doses among the radon-exposed miners, predicted that approximately 14 per cent of lung cancer deaths among such home-residents (about 13,300 deaths per year, or 10 per cent of all U.S. lung cancer deaths) may be due to indoor radon exposure. The 95 per cent confidence intervals are 7-25 per cent or approximately 6,600 to 24,000 lung cancer deaths per year. The attributable risks, i.e., the excess lung cancer rate in the United

States in a population due to exposure to radon progeny as a fraction of the lung cancer rate, are similar for males and females and for smokers and nonsmokers. However, higher baseline risks of lung cancer result in much larger numbers of radon-attributable cancer deaths among males (approximately 9,000 lung cancer deaths per year) and among smokers (approximately 11,000 deaths per year). Since the radon concentration in single family dwellings appears to be log-normally distributed (Nero et al, 1986), because of the apparent skewness of the exposure distribution most of the contribution to the attributable risks arises from exposure rates which exceed 150 Bq m^{-3} (4 pCi L^{-1}); this represents approximately 8 per cent of homes. The models predict that the total annual lung cancer burden in the United States would decrease by 4 to 5 per cent, or by about 3,800 lung cancer deaths per year, if indoor levels were reduced to 150 Bq m^{-3} (4 pCi L^{-1}). This is in contrast to a maximum reduction of lung cancer deaths of 14 per cent if all indoor radon exposure were eliminated (Lubin and Boice, 1989).

A satisfactory method of treating the modifying factor of smoking in lung cancer risk assessment and of establishing levels for protecting the health of the public have not as yet been developed. Thus, the precise radon-induced lung cancer risk in the nonsmoker population is uncertain and the overall effectiveness of mitigating the protective measures for reducing radon exposure in indoor air remains in doubt. Based on the available information, however, the radon risk to the nonsmoker appears to be much less than has been presently estimated. Protective measures are likely to be most effective in reducing radon risk to smokers, who are already at very high risk for lung cancer.

CONCLUSIONS

The BEIR IV Committee (National Research Council, 1988) provided extensive dose-response modeling and statistical fitting, as well as lifetime risk projections for lung cancer mortality following exposure to radon and radon progeny. However, the Committee cautioned that

the risk estimates derived from the epidemiologic data should not be considered as precise numerical values. All were derived from analyses of incomplete human data and involved numerous uncertainties requiring assumptions concerning the factors that modify the carcinogenic risk of exposure to radon progeny. It is expected that these lung cancer mortality risk estimates will change as new information becomes available and methods for analysis continue to improve. The present need to apply risk projections from surveys of underground miners to estimate the lung cancer risk to the general population from exposure to indoor radon introduces numerous additional uncertainties and technical difficulties. The domestic environment has not, as yet, been characterized adequately in terms of the variables affecting the dose and risk from radon progeny. Variations in indoor radon levels, alterations of aerosol characteristics, and impacts of active and passive smoking and nonsmoking risk factors suggest that health consequences of indoor radon exposure require more epidemiologic study and basic research. Studies of the interactions of lung cancer, smoking and exposure to radon in indoor air and in underground mines are required, provided such studies have sufficient statistical power to quantify differences between the risks in the occupational and domestic environmental settings. This will permit assessment of the magnitude of the potential lung cancer risk to the general public from exposure to radon progeny in indoor domestic environments, and thereby help place into perspective the potential carcinogenic effects of radon exposure as an environmental hazard.

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LAWRENCE BERKELEY LABORATORY
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BERKELEY, CALIFORNIA 94720